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The relationship between helicobacter pylori infection and iron deficiency anemia in Yemeni adults

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Abstract

Background: Iron deficiency anemia (IDA) represents a common nutritional and hematological disorder. Helicobacter pylori (H. pylori), primarily recognized for its role in gastritis, peptic ulcers, and gastric cancer, has recently demonstrated a notable correlation with IDA according to emerging research. This study aims to evaluate the potential relationship between H. pylori infection and the development of IDA.
Materials and Methods: This case-control study included 100 participants between the ages of 18-56. The study group consisted of 50 individuals with confirmed H. pylori infection, while the control group comprised 50 healthy subjects. We conducted analyses of stool specimens for H. pylori antigens and occult blood. Additionally, we collected blood samples to evaluate complete blood counts and iron profiles using standardized automated systems.
Results: Our analysis revealed no statistically significant variations in demographic characteristics or risk factors between the study and control groups. The majority of participants (90
Conclusion: H. pylori infection does not demonstrate a significant association with iron deficiency anemia. Key indicators including hemoglobin levels, serum iron, ferritin, TIBC, and UIBC remained statistically comparable between H. pylori-infected participants and healthy controls.

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1. INTRODUCTION

Helicobacter pylori, a Gram-negative bacterium, is a global public health concern. This pathogen disseminates via the fecal-oral transmission route and is associated with chronic gastric inflammation in a substantial proportion of those infected [1]. The prevalence of H. pylori infections is notably higher in pediatric and adult populations in developing nations [2] . Approximately 50% of the global population harbors H. pylori [3, 4], which has the potential to induce a range of gastric pathologies, such as gastritis, gastric ulcers [5], and even more severe conditions, including gastric cancer and lymphoma [1]. Furthermore, H. pylori infection may contribute to a spectrum of health issues that extend beyond the gastrointestinal tract, including iron deficiency anemia [6], hypercholesterolemia [7], thrombocytopenia [8], dyslipidemia [9], cardiovascular diseases, cerebrovascular accidents, and neurological disorders [4]. Numerous scholarly investigations have elucidated a substantial correlation between H. pylori infection and iron deficiency anemia (IDA) [10, 11, 12, 13, 14, 15, 16]. Therapeutic interventions targeting H. pylori infection have been demonstrated to ameliorate IDA in a considerable number of patients [12, 14, 16, 17]. This observation suggests that H. pylori infection may be implicated in the etiology of IDA. The precise biological mechanisms through which H. pylori infection induces IDA remain a

subject of ongoing research. Several hypotheses have been posited: H. pylori has the potential to induce hemorrhagic ulcers and gastritis, resulting in the depletion of iron levels, and may diminish gastric acidity and vitamin C concentrations, both of which are critical for optimal iron absorption. Additionally, it may sequester iron for its own metabolic processes, exacerbating iron deficiency [18, 19, 20]. The incidence of H. pylori infection in Yemen is notably elevated, with an 82% prevalence reported in adult patients presenting with dyspepsia [21], and 65% in symptomatic pediatric populations [22, 23]. Data pertaining to the relationship between iron deficiency anemia and H. pylori infection in the Yemeni context are limited. Consequently, the present study sought to elucidate the association between H. pylori infection and iron deficiency anemia among Yemeni adults.

2. METHODS

2.1. Experiment design and study population

This research constituted a case-control investigation conducted from October 2023 to August 2024. The study took place in Sana'a at three medical facilities: the Clinical Gastroenterology Outpatient Department of the Republican Teaching Hospital, the AL-Ertiqaa Academic Specialized Hospital, and the University of Science and Technology Hospital. The sample population consisted of 100 Yemeni adults aged 18-56 years. Participants were divided into two equal groups: 50 individuals with confirmed H. pylori infection and 50 healthy individuals matched for age and sex who served as controls. The research protocol excluded individuals who had taken NSAIDs (nonsteroidal anti-inflammatory drugs), proton pump inhibitors, H2-receptor antagonists, antibiotics, or iron supplements within the previous four weeks. Furthermore, pregnant women and those experiencing menorrhagia were excluded from participation. All participants underwent an occult blood test (One Step Fecal Occult Blood Test Device; Abon Biopharm, China) to exclude the presence of gastric and duodenal bleeding ulcers. In compliance with ethical standards, the research team obtained approval from the Ethical Committee of Research at the Faculty of Medicine and Health Sciences, Sana'a University, as well as from the Republican Teaching Hospital Authority and the National Center of Public Health Laboratories before initiating the study. Each participant provided written informed consent prior to enrollment.

2.2. Specimen collection and testing

Stool samples were collected from each participant using a plastic container. These samples were analyzed for H. pylori antigen detection within two hours of collection. A one-step testing device (ACON Bio Pharma, China) was utilized for qualitative detection of H. pylori antigen in stool samples. This test employed chromatographic immunoassay methodology, with results available within 10 minutes. Venous blood samples (approximately 5 ml) were collected from each participant and divided into two portions: 2 ml in an EDTA tube for Full Blood Count (FBC) analysis using the DYMIND DF55 Auto 5-part Hematology analyzer (China), and 3 ml in a plain tube for serum separation. The Beckman Coulter AU 480 (United States) automated analyzer was used to measure serum levels of iron, ferritin, total iron binding capacity (TIBC), and unsaturated iron binding capacity (UIBC).

2.3. Data Analysis

Data were generated, organized, encoded, and analyzed the Statistical Package for Social Sciences (SPSS version 20, Chicago, USA). Qualitative data are displayed and expressed in numerical form and percentages. The findings are articulated as mean $\pm$ SD for variables that adhered to a normal distribution, and as geometric mean accompanied by 95% confidence intervals subsequent to the application of log 10 transformation for variables that did not conform to a normal distribution. The relationship between sociodemographic characteristics, risk factors, and H. pylori infection was assessed using the chi-squared test. Means were analyzed using an independent sample t-test. Differences were considered statistically significant when the P-value was less than 0.05.

3. RESULTS

3.1. Sociodemographic features and risk factors for H. pylori infection

The present investigation included a total of 100 participants: 50 with confirmed Helicobacter pylori infection and 50 healthy controls. Table 1 presents the sociodemographic characteristics and risk factors associated with H. pylori infection in our study population. Age distribution analysis showed that 18% of infected cases and 14% of controls were under 21 years old, while 62% of cases and 78% of controls were between 21-40 years old, and 20% of cases versus 8% of controls were between 41-60 years old. Regarding gender distribution, males represented 22% of infected cases (11 participants) and 28% of controls (14 participants), while females accounted for 78% of cases (39 participants) and 72% of controls (36 participants), showing a higher prevalence of H. pylori infection among female participants. Additionally, our findings indicated that 68% of H. pylori infections were reported in urban areas. Statistical analysis using the chi-square test revealed no significant differences in sociodemographic characteristics or risk factors between the two groups (Table (1)).

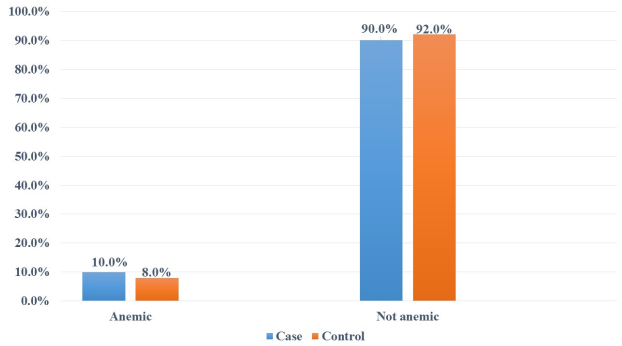

Figure 1. Frequency of anemia among H. pylori infected patients and control.

3.2. Hematological parameters

Analysis revealed that 45 patients (90%) and 46 controls (92%) exhibited normal hemoglobin levels, while anemia was present in 5 patients (10%) and 4 controls (8%), as illustrated in Figure (1). Table (2) presents the comprehensive hematological parameters for H. pylori-infected individuals and the control group. Statistical analysis indicated no significant variations between the two cohorts.

3.3. Iron profile

As depicted in Figure (2), reduced ferritin concentrations (below 30 ng/ml) were identified in 13 H. pylori-infected patients (26%) compared to 9 controls (18%). Although serum iron and ferritin measurements were marginally reduced in H. pylori-infected patients compared to controls, these differences did not reach statistical significance. Conversely, both Total Iron Binding Capacity (TIBC) and Unsaturated Iron Binding Capacity (UIBC) showed slight elevations in patients compared to controls, though these differences also remained statistically non-significant (Table (3)).

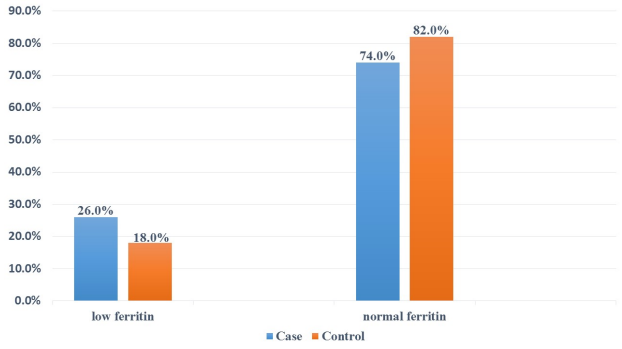

Figure 2. Percentage of ferritin levels among H. pylori infected patients and control.

4. DISCUSSION

The relationship between Helicobacter pylori (H. pylori) infection and iron deficiency anemia (IDA) warrants examination due to H. pylori's potential influence on iron absorption and metabolism. Approximately 50% of the global population is estimated to harbor H. pylori infection [24]. Prior epidemiological research has identified H. pylori infection as a potential contributor to IDA [23, 25]. This investigation sought to examine the correlation between Helicobacter pylori infection and IDA. Our analysis revealed no statistically significant differences in hemoglobin (Hb) levels, red blood cells (RBC), hematocrit (Hct), red blood cell indices (MCV, MCH, and MCHC), red cell distribution width (RDW-CV), white blood cells (WBC), or platelets between study groups. However, applying the World Health Organization's criteria for anemia - hemoglobin levels below 13 g/dL in males and 12 g/dL in females [26], we noted that 10% of H. pylori-infected subjects presented with anemia, compared to 8% in the non-infected control group. These results align with previous studies indicating no significant difference in average levels between H. pylori-infected and non-infected individuals Data are presented as mean ± SD, * Data are presented as geometric mean (95% confidence interval[10, 23, 27] . In contrast, another study demonstrated a statistically significant variation in hemoglobin levels between H. pylori-positive and negative groups [28]. The observed reduction in hemoglobin levels was attributed to gastric atrophy in elderly patients rather than H. pylori infection itself [28]. Our study participants did not exhibit gastric bleeding, and the relationship between H. pylori infection and lower hemoglobin levels might also be explained by gastric hemorrhage [29] Research from two previous studies showed that MCV, MCH, and MCHC levels did not differ significantly between individuals with and without H. pylori infection [28, 24]. Similar findings regarding MCV and MCH have been reported in Palestinian populations [30] . Our results showed reduced serum iron levels, decreased ferritin levels, and increased total iron-binding capacity (TIBC) and unsaturated iron-binding capacity (UIBC) compared to healthy controls; however, these differences were not statistically significant. These findings align with those of Shih et al. in 2013, who reported similar outcomes for serum iron, ferritin, and TIBC [27]. Additionally, a Korean study found no significant differences in serum iron and TIBC between groups with positive and negative H. pylori infections, though a notable decrease in ferritin levels was observed in patients with H. pylori [31]. We disagree with the findings of a previous meta-analysis covering 15 epidemiological studies that identified a significant association between H. pylori infection and iron deficiency anemia in children under 11 years [11]. This strong association was not found in adult populations, suggesting that H. pylori may not significantly contribute

Table 1. Sociodemographic features and risk factors of the study population.

Risk factor	Cases (50)		Controls (50)		Statistical analysis	
	No	%	No	%	χ^2	P
Age groups (years)						
≤ 20	9	18	7	14	3.7	0.15
21-40	31	62	39	78		
41-60	10	20	4	8		
Gender						
Males	11	22	14	28	0.48	0.49
Females	39	78	36	72		
Marital status						
Single	20	40	22	44	0.16	0.69
Married	30	60	28	56		
Residence						
Rural	16	32	14	28	0.19	0.66
Urban	34	68	36	72		
Smoking status						
Yes	5	10	4	8	0.12	0.73
No	45	90	46	92		
Family history						
Yes	26	52	21	42	1.004	0.32
No	24	48	29	58		
Sewage system						
Available	25	50	33	66	2.6	0.11
Not available	25	50	17	34		
Water source						
Tap	42	84	43	86	1.3	0.52
Non-refined	8	16	7	14		
Personal hygiene						
Yes	34	68	38	76	0.79	0.37

χ^2:chi square,p:p-value

Table 2. Hematological parameters among H. pylori infected patients and control.

Parameters	H. pylori patients (n = 50)	Controls (n = 50)	p-value
Hb (g/dL)	13.9 ± 1.6	14.2 ± 1.9	0.38
RBCs ($\times 10^{12}$/L)	5 ± 0.6	5 ± 0.5	0.85
Hct (%)	41.3 ± 4.6	41.6 ± 4.9	0.74
MCV (fL)	83.3 ± 6.3	82.6 ± 9.6	0.64
MCH (pg)	28 ± 2.3	28.3 ± 3.0	0.54
MCHC (g/dL)	33.5 ± 1.5	33.6 ± 1.4	0.79
RDW-CV (%)	14 ± 1.3	13.4 ± 1.6	0.54
WBCs ($\times 10^9$/L)	6.4 ± 2.1	5.8 ± 1.7	0.09
PLTs* ($\times 10^9$/L)	296 ± 86.3	325 ± 63.5	0.06

Data are presented as mean ± SD evaluated by the independent-samples t test.
WBC, white blood cell count; RBC, red blood cell count; Hb, hemoglobin; Hct, hematocrit
MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration
RDW, mean red cell distribution width; PLT, platelet count
SD, standard deviation. p: p-value.

to iron-deficiency anemia in adults as it does in children and adolescents [11]. Most participants in our study were adults. While many cross-sectional studies have reported a significant connection between reduced iron stores, iron deficiency anemia (IDA), and H. pylori infection prevalence in pediatric populations, other studies have not confirmed this relationship [32, 33, 34, 35]. Our findings conflict with a previous report indicating that individuals with H. pylori infection showed significantly lower serum iron, higher TIBC, and increased serum ferritin levels compared to the control group [36]. In our study, 13 patients (26%) and nine controls (18%) were identified as having iron deficiency, defined as ferritin levels below 30 ng/mL [37]. The low prevalence of anemia and iron deficiency in this study makes it difficult to clarify the relationship between H. pylori infection, anemia, and iron deficiency in our participants. There are reasonable explanations for the connection between H. pylori infection, iron deficiency (ID), and iron deficiency anemia (IDA), though the underlying mechanisms remain unclear. H. pylori infection may reduce gastric ascorbic acid levels and serum iron concentrations, potentially

Table 3. Iron profile among H. pylori infected patients and control.

Parameters	H. pylori patients (n = 50)	Control (n = 0)	p-value
*Serumiron** (µmol/L)	11.3(9.9-13.0)	11.7(10.5-13.1)	0.66
Serum ferritin* (ng/ml)	44.3(34.3-57.1)	48.2(40.2-57.8)	0.59
TIBC (µmol/L)	58.0 ± 8.1	56.8 ± 9.7	0.51
UIBC (µmol/L)	45.3 ± 8.9	44.4 ± 10.1	0.65

Data are presented as mean ± SD, * Data are presented as geometric mean (95% confidence interval) evaluated by the independent-samples t-test. Data are presented as mean ± SD evaluated by the independent-samples t-test. TIBC, total iron-binding capacity; UIBC, unsaturated iron-binding capacity; SD, standard deviation. p: p-value.

inhibiting iron absorption [38, 39, 15]. Additionally, H. pylori's colonization of the stomach requires ongoing iron intake to support bacterial growth, which may deplete iron reserves [40]. This colonization affects the gastric mucosa, potentially resulting in decreased iron absorption due to hypochlorhydria and chronic gastritis [41]. Another mechanism contributing to iron deficiency and IDA is blood loss associated with ulcerative gastritis [42]. The differences between our results and those of other studies may be attributed to the specific characteristics of our study populations (including geographic and ethnic variations), sample sizes, sampling methods, and diagnostic techniques used to identify H. pylori infection. Furthermore, our case-control design limits our ability to establish cause-and-effect relationships. Selection bias was present because our participants were recruited from gastroenterology clinics.

5. CONCLUSION

This study revealed no significant correlation between H. pylori infection and iron deficiency anemia among Yemeni adults. Nevertheless, to validate these conclusions and investigate underlying mechanisms, additional studies employing larger cohorts and longitudinal methodologies are essential. We observed that iron profiles and hematologic parameters remained comparable between H. pylori-infected individuals and the control group. Considering the ongoing debate surrounding the relationship between H. pylori infection and iron deficiency anemia, further investigation into the potential mechanisms that may contribute to iron-deficiency anemia in H. pylori-infected individuals is necessary.

DISCLOSURE

All authors have no conflicts of interest to declare.

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REFERENCES

[1] Eugène Tshibangu-Kabamba and Yoshio Yamaoka. "Helicobacter pylori infection and antibiotic resistance—From biology to clinical implications". In: *Nat. Rev. Gastroenterol. & Hepatol.* 18.9 (2021), pp. 613–629. DOI: 10.1038/s41575-021-00449-x.

[2] Kallirroi Kotilea, Patrick Bontems, and Eliette Touati. "Epidemiology, diagnosis and risk factors of Helicobacter pylori infection". In: *Adv. Exp. Med. Biol.* 1149 (2019), pp. 17–33. URL: https://pasteur.hal.science/pasteur-02868133v1.

[3] James KY Hooi et al. "Global prevalence of Helicobacter pylori infection: Systematic review and meta-analysis". In: *Gastroenterology* 153.2 (2017), pp. 420–429. DOI: 10.1053/j.gastro.2017.04.022.

[4] Antonietta Gerarda Gravina et al. "Helicobacter pylori and extragastric diseases: A review". In: *World J. Gastroenterol.* 24.29 (2018), p. 3204. DOI: 10.3748/wjg.v24.i29.3204.

[5] Anca Laura Dincă, Lorena Elena Meliț, and Cristina Oana Mărginean. "Old and new aspects of *H. pylori*-associated inflammation and gastric cancer". In: *Children* 9.7 (2022), p. 1083. DOI: 10.3390/children9071083.

[6] Claudiu Dan Mărginean, Cristina Oana Mărginean, and Lorena Elena Meliț. "Helicobacter pylori-related extraintestinal manifestations—myth or reality". In: *Children* 9.9 (2022), p. 1352. DOI: 10.3390/children9091352.

[7] Abduselam Abdu et al. "Dyslipidemia and associated factors among patients suspected to have Helicobacter pylori infection at Jimma University Medical Center, Jimma, Ethiopia". In: *Int. J. Gen. Med.* 13 (2020), pp. 311–321. DOI: 10.2147/IJGM.S243848.

[8] Germán Campuzano-Maya. "Hematologic manifestations of Helicobacter pylori infection". In: *World J. Gastroenterol.* 20.36 (2014), p. 12818. DOI: 10.3748/wjg.v20.i36.12818.

[9] Mohammed Hashim, Omar Mohammed, and Meseret Wolde. "The association of Helicobacter pylori infection with dyslipidaemia and other atherogenic factors in dyspeptic patients at St. Paul's Hospital Millennium Medical College". In: *Heliyon* 8.5 (2022). URL: http://creativecommons.org/licenses/bync-nd/4.0/.

[10] Shunji Kato, Benjamin D Gold, and Atsushi Kato. "Helicobacter pylori-associated iron deficiency anemia in childhood and adolescence-pathogenesis and clinical management strategy". In: *J. Clin. Med.* 11.24 (2022), p. 7351. DOI: 10.3390/jcm11247351.

[11] Xinhua Qu, Xiaoli Huang, Ping Xiong, et al. "Does Helicobacter pylori infection play a role in iron deficiency anemia? A meta-analysis". In: *World J. Gastroenterol.* 16.7 (2010), pp. 886–896. DOI: 10.3748/wjg.v16.i7.886.

[12] Lajos Hudak et al. "An updated systematic review and meta-analysis on the association between Helicobacter pylori infection and iron deficiency anemia". In: *Helicobacter* 22.1 (2017), e12330. DOI: 10.1111/hel.12330.

[13] Yasser A Rahman et al. "Helicobacter pylori and its hematological effect". In: *Egypt. J. Intern. Med.* 31.3 (2019), pp. 332–342. DOI: 10.4103/ejim.ejim_103_18.

[14] Jeevan M Saju et al. "Is Helicobacter pylori a reason for unexplained iron deficiency anemia: a systematic review". In: *Cureus* 14.9 (2022). DOI: 10.7759/cureus.29112.

[15] Nada Mubarak et al. “Helicobacter pylori, anemia, iron deficiency and thrombocytopenia among pregnant women at Khartoum, Sudan”. In: *Trans. Royal Soc. Trop. Med. Hyg.* 108.6 (2014), pp. 380–384. DOI: 10.1093/trstmh/tru044.

[16] Beatrice Boutamba Eyoum Bille and Laure Brigitte Kouitcheu Mabeku. “Relationship between active Helicobacter pylori infection and anemia, iron deficiency, iron deficiency anemia: a cross-sectional study in a sub-Saharan setting”. In: *JGH Open* 6.8 (2022), pp. 554–568. DOI: 10.1002/jgh3.12787.

[17] Suhair N Mwafy and Wesam M Afana. “Hematological parameters, serum iron and vitamin B 12 levels in hospitalized Palestinian adult patients infected with Helicobacter pylori: A case–control study”. In: *Hematol. Transfus. Cell Ther.* 40.2 (2018), pp. 160–165. URL: http://creativecommons.org/licenses/by-nc-nd/4.0/.

[18] Feng-Woei Tsay and Ping-I Hsu. “*H. pylori* infection and extra-gastroduodenal diseases”. In: *J. Biomed. Sci.* 25.1 (2018), pp. 1–8. DOI: 10.1186/s12929-018-0469-6.

[19] Johanna Andrea Urrego Diaz, William Otero Regino, and Mario Gomez Zuleta. “Helicobacter pylori and hematologic diseases”. In: *Revista Colombiana de Gastroenterol.* 28.4 (2013), pp. 324–331. URL: http://www.scielo.org.co/pdf/rcg/v28n4/en_v28n4a08.pdf.

[20] Jonathon Snook, Neeraj Bhala, Ian LP Beales, et al. “British Society of Gastroenterology guidelines for the management of iron deficiency anaemia in adults”. In: *Gut* 70.11 (2021), pp. 2030–2051. DOI: 10.1136/gutjnl-2021-325210.

[21] Ahmed A Gunaid, Nabil A Hassan, and Iain Murray-Lyon. “Prevalence and risk factors for Helicobacter pylori infection among Yemeni dyspeptic patients”. In: *Saudi Med. J.* 24.5 (2003), pp. 512–517. URL: https://d1wqtxts1xzle7.cloudfront.net/43723590/Prevalence_and_risk_factors_for_Helicoba20160314-12081-120kbyg-libre.

[22] Khitam Muhsen and Dani Cohen. “Helicobacter pylori infection and iron stores: a systematic review and meta-analysis”. In: *Helicobacter* 13.5 (2008), pp. 323–340. DOI: 10.1111/j.1523-5378.2008.00617.x.

[23] SA Al-Ashwal et al. “Levels of Immunoglobulins among iron deficiency anemic children in Sana’a City Yemen”. In: *Sana’a Univ. J. Med. Health Sci.* 17.2 (2023). DOI: 10.59628/jchm.v17i2.411.

[24] N Miguel. *Hematological extradigestive manifestations of helicobacter pylori infection in childhood*. 2016. DOI: 10.5772/63827.

[25] Nils Milman et al. “Serum ferritin, hemoglobin and Helicobacter pylori infection: A seroepidemiologic survey comprising 2794 Danish adults”. In: *Gastroenterology* 115.2 (1998), pp. 268–274. URL: https://www.gastrojournal.org/action/showPdf?pii=S0016-5085%5C%2898%5C%2970192-1.

[28] Hsiu-Yun Shih et al. “Helicobacter pylori infection and anemia in Taiwanese adults”. In: *Gastroenterol. Res. Pract.* 2013.1 (2013), p. 390967. DOI: 10.1155/2013/390967.

[29] Sang Yong Lee et al. “Low Hemoglobin Levels are Related to the Presence of Gastric Atrophy Rather Than the Presence of *H. pylori* Infection Itself: A Study of 2,398 Asymptomatic Adults”. In: *Gastroenterology* 142.5 (2012), S–474. URL: https://www.gastrojournal.org/article/S0016-5085(12)61799.

[26] Dulciene Maria Magalhães Queiroz, Paul R Harris, Ian R Sanderson, et al. “Iron status and Helicobacter pylori infection in symptomatic children: An international multi-centered study”. In: *PLoS One* 8.7 (2013), e68833. DOI: 10.1371/journal.pone.0068833.

[27] World Health Organization. *Nutritional anaemias: report of a WHO scientific group*. Tech. rep. World Health Organization, 1968.

[30] Karen Miernyk, Dana Bruden, Carolyn Zanis, et al. “The effect of Helicobacter pylori infection on iron stores and iron deficiency in urban Alaska Native adults”. In: *Helicobacter* 18.3 (2013), pp. 222–228. DOI: 10.1111/hel.12036.

[31] Y Dogan et al. “Lactoferrin levels in the gastric tissue of Helicobacter pylori positive and -negative patients and its effect on anemia”. In: *Mediat. Inflamm.* 2012.1 (2012), p. 214581. DOI: 10.1155/2012/214581.

[32] JY Lee et al. “Helicobacter pylori infection and iron deficiency in non-elderly adults participating in a health check-up program”. In: *Korean J. Intern. Med.* 37 (2022), pp. 304–312. DOI: 10.3904/kjim.2020.433.

[33] JW Choi. “Does Helicobacter pylori infection relate to iron deficiency anemia in prepubescent children less than 12 years of age?” In: *Acta Paediatr.* 92.8 (2003), pp. 970–972. DOI: 10.1111/j.1651-2227.2003.tb00633.x.

[34] MT Haghi-Ashtiani et al. “Anemia in children with and without Helicobacter pylori infection”. In: *Arch. Med. Res.* 39.5 (2008), pp. 536–540. DOI: 10.1016/j.arcmed.2008.04.005.

[35] IS Santos et al. “Helicobacter pylori is not associated with anemia in Latin America: results from Argentina, Brazil, Bolivia, Cuba, Mexico and Venezuela”. In: *Public Health Nutr.* 12.10 (2009), pp. 1862–1870. DOI: 10.1017/S1368980009004789.

[36] G Sandström et al. “Helicobacter pylori Antibodies and Iron Deficiency in Female Adolescents”. In: *PLoS One* 9.11 (2014), e113059. DOI: 10.1371/journal.pone.0113059.

[37] G Kishore et al. “Association between Helicobacter pylori infection and serum iron profile”. In: *Cureus* 13.9 (2021). DOI: 10.7759/cureus.17925.

[38] C Camaschella. “Iron-deficiency anemia”. In: *New Engl. J. Med.* 372 (2015), pp. 1832–1843. DOI: 10.1056/NEJMra1401038.

[39] M Öztekin et al. “Overview of Helicobacter pylori infection: clinical features, treatment, and nutritional aspects”. In: *Diseases* 9.4 (2021), p. 66. DOI: 10.3390/diseases9040066.

[40] Y Essadni et al. “Iron Deficiency Anemia: The Contribution of Upper Digestive Endoscopy in Etiological Investigation”. In: *Saudi J. Med. Pharm. Sci.* 10.2 (2024), pp. 120–124. DOI: 10.36348/sjmps.2024.v10i02.010.

[41] BR Otto, AMJJ Verweij-van Vught, and DM Maclaren. “Transferrins and Heme-Compounds as Iron Sources for Pathogenic Bacteria”. In: *Crit. Rev. Microbiol.* 18 (1992), pp. 217–233. DOI: 10.3109/10408419209114559.

[42] H Monzón et al. “Helicobacter pylori infection as a cause of iron deficiency anaemia of unknown origin”. In: *World J. Gastroenterol.* 19.26 (2013), pp. 4166–4171. DOI: 10.3748/wjg.v19.i26.4166.